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HEARING LOSS IN DECOMPRESSION

by

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Bureau of Medicine and Surgery, Navy Department

Research Work Unit MF12.524.004-9012D.02

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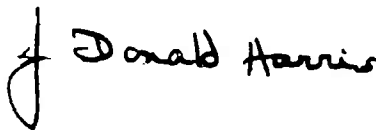
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SUBMARINE MEDICAL RESEARCH LABORATORY  
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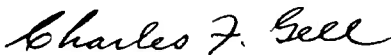
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## **SUMMARY PAGE**

### **THE PROBLEM**

To review the possible etiopathology of sudden deafness in divers undergoing decompression.

### **FINDINGS**

Only a few of the many causes of sudden deafness familiar to all otologists seem to contribute to the occasional loss of hearing encountered during the decompression phase of a real or simulated underwater excursion. Decompression itself is implicated in ten case histories as a probable causative agent, either by way of microhemorrhage or microembolism.

### **APPLICATION**

This report should serve as a background bibliography and a start on the problems of preventing and treating decompression hypacusis. Much more oto-audiologic data on future patients should be collected than is currently generally the case: it is considered that a few audiograms showing the progress of recovery of hearing are insufficient to help to distinguish the site and manner of injury.

### **ADMINISTRATIVE INFORMATION**

This investigation was conducted as part of Bureau of Medicine and Surgery Work Unit MF12.524.004-9012D—Physiological Psychology of the Ear Under Stress. The present report is No. 2 on this Work Unit. It was approved for publication on 5 August 1969, and designated as Submarine Medical Research Laboratory Report No. 591.

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## ABSTRACT

Among those who are exposed repeatedly to compression-decompression cycles, a relatively high incidence of otologic symptoms including hearing loss is often reported. Most of this can readily be attributed to the residual effects of repeated aerotitis media, and is not sudden. A review of the topic of sudden deafness reveals that every large hospital where such records have been published sees every month about 1-2 patients whose sudden deafness is not easily explained. Possible causes have been suggested: acute neuritis of the VIIIth nerve, virus infection, vascular accident, vasomotor neurosis, acoustic trauma at levels of noise not usually noxious, collagen disease, transient ischemia from violent exercise or strong emotion, or from alterations of the cervical spinal column.

None of these causes, however, would seem to underlie a series of patients experiencing sudden hearing loss of a moderate or often profound level during decompression, and which may respond favorably to immediate recompression. Ten patients with hearing loss under decompression are presented in the Appendix, two of whom recovered their hearing loss completely during recompression, and three with only slight recovery. It seems likely that these five patients had sustained bubble formation in one or more branches of the internal acoustic artery. In the five patients not responding to recompression, but treated with decongestants, vasodilators, etc., the possibility also exists of microhemorrhage in the cochlea. A brief summary is given of current therapeutic regimens.

## HEARING LOSS IN DECOMPRESSION

The structures of the ear are so delicately formed and balanced that we are sensitive to acoustic stimuli over a very broad range of 10 octaves in frequency and to pressures near 0.0002 dynes/cm<sup>2</sup>, at which value particle movements of the air are as minute as 10<sup>-10</sup> cm. Yet over and above this extreme sensitivity the ear can support acoustic pressures a billion times greater, or 140 db. But even this does not exhaust the elasticity of the ear, for the middle ear at least can undergo static displacements, as in the modified Valsalva maneuver of an order of magnitude greater than are found in response to intense sound.

It is only because of these large safety factors and pressure release mechanisms that we are enabled to withstand the innumerable severe insults to our auditory mechanism which are the everyday lot of the diver. The fact is that a corps of men can be formed by sharp selection and training which can continue to perform well for years while undergoing the constant auditory rigours and dangers of a diving career. Shilling and Everley<sup>69</sup> show that the mean audiogram of divers who have had no special exposure to intense noise was not appreciably worse than controls; Coles<sup>14</sup> examined 57 very experienced divers, and found their hearing perfectly normal except for those who had experienced "much" gunfire or small-arms noise. Evidently in a cadre of men who do not experience barotrauma and who are protected by proper procedures from bubble formation, hearing may remain perfectly normal. There is thus no suggestion that repeated compression-decompression cycles per se have any effect on auditory acuity.

There are, however, many individuals for whom pressure equalization across the drum is not semi-automatic, or even possible, and there are fairly frequent occasions when even experienced divers find it difficult for one reason or another to "clear" their ears or to do so with sufficient rapidity. Furthermore, there are those who have experienced sudden hearing losses during a dive, of which the etiology is still obscure, though thought to

be associated in some way with their diving experience. It is with these two types of cases that we deal in this paper.

### A. Aerotitis Media.

Armstrong and Heim<sup>2</sup> thoroughly explored the condition they termed aero-otitis media, caused by a lack of ventilation of the middle ear. All attending physicians must have seen through the otoscope the drastic changes which can sometimes occur in the ear drum and lining of the tympanic cavity, with blood coming from the Eustachian tube. The classification by Teed<sup>75</sup> into Categories 1-5 of increasing damage is well known. From this laboratory Shilling and Everley reported a series of 2,751 submariners, only nine of whom finally failed to pass the 50lb pressure test. In their series, 90% of men taking the test for the first time required no change in pressurization rate to completion, while 91.5% of those who had passed the test at least once required no change.

The condition is worse with air than with helium-oxygen mixtures because of the increased speed with which HeO<sub>2</sub> can diffuse through the Eustachian tube. Thus, in acute conditions, Lovelace, et al,<sup>47</sup> Crosson, et al,<sup>18</sup> Hall,<sup>30</sup> and Requarth,<sup>62</sup> found that caisson workers could obtain relief, reenter a pressure chamber, and suffer less infection and suppuration if the pressure change was performed in HeO<sub>2</sub>.

Haines and Harris<sup>29</sup> performed otoscopy, nasopharyngoscopy, and pre- and post-pressure audiometry on 6,149 men. Almost no effect on hearing was found unless the middle ear contained serosanguinous fluid. Alfandre<sup>1</sup> in another series of 432 men from this laboratory generally confirms these conclusions.

Occasionally, even an experienced diver will experience an ear "squeeze," a more or less severe case of aerotitis media, as when there is a temporary catarrhal obstruction of the Eustachian tube or if the rate of descent is too fast, as in a free fall. Most eardrums will then rupture at about a half an atmosphere of difference across the drum. But when such damage occurs, and heals, and the eardrum is

scarred, it is never quite as strong again, and may rupture at much less than a half an atmosphere of pressure. Coles<sup>14</sup> mentions one case of a very thin scar tissue which ruptured at a pressure differential the equivalent of six feet. Such a person would be quickly screened out of a diving cadre.

#### B. Sudden Deafness of Obscure Origin.

An early case of obscure origin was mentioned in 1922 by Kobrak, and since then hundreds of cases have been fully described. Noteworthy, are the papers of DeKleyn<sup>18</sup> (N:21), Rasmussen<sup>61</sup> (N:14), Fowler<sup>26</sup> (N:26), Opheim<sup>58</sup> (N:3), Lindsay and Zuidema<sup>46</sup> (N:6), Hilger and Goltz<sup>82</sup> (N:14), Sacher<sup>63</sup> (N:5), Hallberg<sup>31</sup> (N:178), Bocca and Giordano<sup>7</sup> (N:28), Svane-Knudsen<sup>73</sup> (N:21), Van Caneghem<sup>77</sup> (N:64), Saunders and Lippy<sup>64</sup> (N:9), Boriani<sup>9</sup> (N:10), Wilmot<sup>82</sup> (N:47), Walander and Rubensohn<sup>80</sup> (N:8), Taniewski<sup>74</sup> (N:11), Sheehy<sup>67</sup> (N:223), Bosatra and DeStefani<sup>10</sup> (N:47), Jerger, et al<sup>36</sup> (N:12), Yoshida<sup>84</sup> (N:131), Gontarski, et al<sup>28</sup> (N:11), Yamasaki<sup>83</sup> (N:109), VanDishoeck<sup>78</sup> (N:150), Nozue, et al<sup>56</sup> (N:51), Jokobi and Skurczynski<sup>34</sup> (N:41), Coyas<sup>15</sup> (N:2), Faltiner<sup>21</sup> (N:5), Kessler<sup>40</sup> (N:23), Shiga<sup>68</sup> (N:31), Borasi and Sperati<sup>8</sup> (N:3), Cocks<sup>12</sup> (N:20), Faltynnek and Vesely<sup>22</sup> (N:18), Jaffe<sup>35</sup> (N:143), Lumio and Aho<sup>48</sup> (N:103), Basseres, et al<sup>4</sup> (N:42), Kleinfeldt<sup>42</sup> (N:649).

The observations of Bosatra and DeStefani<sup>10</sup> may be taken to be typical of these surveys: one case experienced bilateral loss after emotion strong enough to redden the face; seven cases after cooling, usually driving with an open window; five cases probably in connection with Asian flu, 34 cases with no clue whatever. Bilateral loss occurred in five of forty-seven patients.

Four types of possible etiology have been suggested by most authors for unexplained sudden deafness.

(1) **Acute neuritis of the VIIIth nerve.** Some patients show an audiologic picture of neurinoma rather than hair cell involvement, usually with vestibular disorders, and in these an allergic or serous neuritis might have occurred. In one clinical case from DeStephani, histology showed an extreme edema interrupting VIIIth nerve fibres at the base of the

modiolus. The edema and disruption may also occur more distally, within the cochlea itself.

(2) **Virus infections.** Van Dishoeck and Bierman<sup>79</sup> proved by immunological research, the strong connection between sudden deafness and virosis. Schuknecht, et al,<sup>65</sup> noted that about 25% of patients with sudden deafness complain of a "cold." In four patients diagnosed as viral labyrinthitis, histology showed, in fact, that the picture was **not** that seen in experimental vascular occlusion in the animal, and it was concluded that a mumps-like virus may be important in sudden deafness. Beal, et al,<sup>6</sup> confirmed this, and stated that the port of entry of the virus particles into the cochlea was via the stria vascularis during viremia. Jaffe<sup>35</sup> reported 40 of 143 patients had viral symptoms. VanDishoeck<sup>78</sup> reported 50 of 153 patients, about 1/3 overall. Jaffe pointed out that virus particles could influence intracochlear blood flow by (1) causing a hypercoagulable state, (2) causing edema of the endothelial cells of the capillaries, narrowing the lumen, and (3) the viral particles attaching to the erythrocytes and causing hemagglutination in vivo, and sludging the blood as seen in the fingernail bed and in the retina. In his cases were five pregnant or post-partum patients and eight post-surgical patients. In both of these situations there is a rise in platelet number and in platelet adhesiveness, either of which, and especially both, may lead to microthrombi in the inner ear. Thus it is likely that the pathogenesis of viral infections leading to sudden deafness is in fact a vascular obstruction to the intracochlear structures. In this connection, one recalls that the capillaries feeding the organ of Corti are actually on the basilar membrane, in intimate association with the stria vascularis rather than further off, as formerly thought.

(3) **Vascular accident.** Many writers have suggested thrombosis, embolism, hemorrhage, or vasospasm due to generalized or localized cardiovascular disease — labyrinthine ischemia. Here the reader should review the cochlear circulation (Perlman and Kimura<sup>59</sup>). For example, a thrombosis of the labyrinthine artery or of the internal acoustic artery immediately after its origin can result

in complete loss of both cochlea and vestibule; a thrombosis of the common cochlear artery will suppress the cochlea, while the vestibular artery will feed the posterior labyrinth; and a thrombosis of the cochlear artery proper will suppress the low tone, but not the high, since the basal spiral also receives from the cochleo-vestibular artery (see several cases in Maurer<sup>52</sup>).

The histological picture is of the hair cells of the cochlea being most susceptible, followed by the nerves, and finally by the vestibule. Thus, more or less transient spasms of the labyrinthine vessels affect first the hair cells, as determined by, for example, the presence of recruitment. In Jerger, et al,<sup>36</sup> series of 12 patients who knew almost the minute their loss occurred, and whose hearing did not subsequently change, four gave flat audiometric losses at 50 db with an audiological test pattern, typical of peripheral organ disorder, while eight gave sloping losses of 60-90 db with test pattern typical of VIIIth nerve lesion. Tetu, et al,<sup>76</sup> offer a series of 33 patients, 14 with confirmed vascular accident and retinal vascular disturbances, and 19 in which they concluded that hemorrhage, paralysis of the vessels, or obstruction by vascular spasm must have caused the hypacusis. They felt their vascular hypothesis was correct since treatment for that condition improved most of the cases. These patients must have been highly selected.

It is not difficult to picture severe and sudden vasometer lesions in the branches of the internal acoustic artery. The vascular bed of the guinea pig has been examined, for example, by Weille,<sup>81</sup> Irwin, et al,<sup>33</sup> and Perlman and Kimura;<sup>59</sup> vascular spasms, stasis, and blood sludge may follow such stressors as CO<sub>2</sub> breathing, cooling, intake of histamine, etc., bringing about alterations in the endolymph and perilymph with probable deleterious effects on the neuroreceptors.

(4) **Vasometer neurosis.** Bosatra and DeStefani<sup>10</sup> advance the interesting hypothesis that a "vasometer neurosis" may underlie much unexplained sudden deafness, much as it may underlie chronic glaucoma and Meniere's disease, by way of a primary alteration of the capillary and precapillary circulation. Such alteration would lead directly to deteri-

oration of structures maintained metabolically, such as hair cells, dendrites, etc. Arnold and Ohsaki<sup>3</sup> point out that blood vessel sludging can stem from disorders in neurovascular regulation. Jakobi and Skurcynski<sup>34</sup> feel indeed that many cases of sudden deafness should be looked on as acute extreme hydrops labyrinthi, neuronegative in nature. They immediately trephined the footplate, reporting nine complete cures in 41 patients; ten more improved and five were able to hear loud speech. This is more than 50% better than is often reported by other treatments. Borasi and Sperati<sup>8</sup> had three skin-divers with unilateral hypacusia, from diving under apnea at 5-10 m. The precipitating causes were pressure imbalance, cold temperatures, hypercapnea and hypoxemia, but the authors felt the etiopathogenesis was a neurovascular angioneurotic mechanism, which yielded within a month in all three cases to anti-edematous drugs and vasodilators. Kleinfeldt,<sup>42</sup> in the largest sample yet published (N:649) concluded that the pathogenesis of the condition is based on an abnormality of the nervous regulation of the smallest blood vessels in the internal ear. It is important here to recall that the cochlear and cochleo-vestibular branches of the internal acoustic artery are "end branches" and there is no collateral circulation.

(5) **Acoustic Trauma.** Reports persist that some episodes of sudden deafness occur in or shortly after exposure to loud noises, but of intensity not such as usually thought noxious. For a review of nine earlier patients, see Kawata and Suga,<sup>38</sup> who had twenty patients over seven years, almost all with flat or U-shaped audios, about half with recruitment, but only two with vestibular symptoms. Faltynek and Vesely<sup>22</sup> present five cases. Kecht<sup>39</sup> had four patients presenting bilateral and partly severe asymmetric inner ear losses; and one total unilateral, to noises of pneumatic drills, circular saws, and a steam engine. This is now termed "akustische unfalles."

(6) **Miscellaneous Suggestions.** Arnold and Ohsaki<sup>3</sup> report a patient with diagnosis of collagen disease, successfully treated with two weeks regime of Prednisone. Mori<sup>154</sup> reports a case from violent physical effort, with

spontaneous recovery in six weeks, apparently a transient ischemia. Kleinfeldt<sup>42</sup> noted that in 16% of his large sample there were alterations of the cervical spinal column. This reminds one of Fields', et al.,<sup>24</sup> demonstration of effects on vestibular function of ischemia due to compression of the vertebral arteries by hypertension of the neck and extreme rotation of the head. Other possibilities include neuritis, secondary to occult diabetes or multiple sclerosis.

In summary, one can say that in many cases the precipitating causes mentioned, such as viremia, exposure to low temperature, loud noises, emotion, sudden physical work, etc., must be overlaid on a previous state of sensitization and abnormal reactivity of the local or general vegetative nervous system.

(7) **Decompression Sickness.** But even after discounting some cases of sudden deafness in a cadre of divers as arising from such causes as discussed heretofore, there remain a number of cases of sudden hearing loss in such a population which seem inescapably to be more directly connected to decompression. We present ten cases in the Appendix, one from our own laboratory and nine from the Experimental Diving Unit, courtesy of Dr. Summit and the EDU staff.

In the case of RC, an open-water diver in our laboratory, two episodes one year apart gave a picture of intracochlear hemorrhage, probably of the internal acoustic artery, producing in the first episode a severe and in the second episode a mild flat loss, but recovering to normal, except for a permanent 15-db dip at 1 kc/s. The nine cases from EDU were all encountered during decompression from dives of 300 ft or more on HeO<sub>2</sub>. The fact that two of these men, H.G.J. and C.R.K., recovered their hearing during recompression almost as suddenly as they lost it, would seem to mean certainly that bubble formation was the sole cause of their defect, with resulting hypoxia or even anoxia in the hair cells. In a similar case, a diver, R.E.J., suffered a sudden profound loss of hearing, which responded only partly to recompression. Most likely bubble formation had led to local hypoxia of sufficient duration to damage permanently the receptor cells.

Of those with residual permanent defects, one (S.) audiogram is U-shaped down a maximum of 45 db at 1-2 kc/s and the other (R. E.J.) is U-shaped down a maximum of 30 db at 500 c/s; while generally flat audiograms are found in the other four men down 40, 50, 70 and 70/80 db. Evidently, if bubble formation is the etiopathology, it is rather general throughout the cochlea.

Of those whose hearing returned substantially during subsequent days or weeks, RC recovered after three weeks, one EDU diver (S.) recovered 25 db after a few weeks and another (M.) after four weeks. From the time course and the extent of recovery, one may conclude that these men are likely to have suffered hemorrhage rather than bubble formation. On the other hand, those men who did not respond to recompression, and whose losses did not return to normal, as in the cases of five EDU divers (R.L.W., W.W.D., R.E.J., T, and B), are likely to have suffered bubble formation.

In cases of sudden hearing loss during decompression, recompression is always performed, though if the etiopathology is a vascular accident such as a spasm, or hemorrhage, it would not be expected to help. Several authors (Gontarski, et al.,<sup>28</sup> Jaffe,<sup>35</sup> Basseres, et al.<sup>4</sup>) advocate immediate hospitalization for every case of sudden deafness, with virologic, vascular, humoral, and audiological examination. A minimum regimen for a cadre of divers would seem to be the taking of reference and even pre-dive audiograms, and a complete battery of virologic, vascular, humoral, and audiological tests after every acute episode. All authors agree that treatment is hopeless if deferred. In a large series Sheehy<sup>67</sup> had 62% recover if treated within four days, only one if treatment was deferred beyond six weeks. Segal,<sup>66</sup> Piesel<sup>60</sup> and Lumio and Aho<sup>48</sup> confirm this. Cocks,<sup>12</sup> Maurer,<sup>52</sup> and Kessler<sup>40</sup> have had good success with stellate ganglion block, if given very early. Kessler<sup>41</sup> performed 610 stellate ganglion blocks on 94 patients over seven years, for Meniere's syndrome, and for sudden loss of hearing, though with three fatal accidents; he cautions against inexperienced use Gailard<sup>27</sup> notes that in the case of a hemorrhage,



an anti-coagulant may obviously do more harm than good. Coyas<sup>15</sup> gives a regime of testing for clotting time to govern dosage of heparin and Sintrom. In cases of supposed ischemia of the labyrinth, Boriani,<sup>9</sup> Faltiner<sup>21</sup> and Faltynek and Vesely<sup>22</sup> report very good success with ATP. The latter present 13 patients with supposed ischemia of the organ of Corti, and five with acute acoustic trauma, but from generally non-noxious levels. Fourteen patients showed complete improvement, three patients showed substantial improvement, one patient showed no improvement. Jaffe<sup>35</sup> gives a summary of a suggested regimen with the most recent drugs. Strange<sup>70</sup> cautions that treatment over weeks is not only justified but necessary, as with some patients he found improvements through the 70th day. He gave, on alternative days, (1) Complamin, Ozothin, Neurobion, and Rovigon, and (2) infusions of 7% NaHCO<sub>3</sub> and stellate ganglion block.

### SUMMARY

In summary, one can say that among those who are exposed repeatedly to compression-decompression cycles, such as aviators, the breath-holding Ama shellfish divers of Japan, military and industrial divers and caisson workers, etc., a relatively high incidence of otologic symptoms including hearing loss is often reported. Most of this can readily be attributed to the residual effects of repeated aerotitis media, and the onset is not sudden. A review of the topic of sudden deafness reveals that every large hospital where such records have been published sees every month one or two patients whose sudden deafness is not easily explained. Possible causes have been suggested: acute neuritis of the VIIIth nerve, virus infection, vascular accident, vasometer neurosis, acoustic trauma at levels of noise not usually noxious, collagen disease, transient ischemia from violent exercise or strong emotion, or from alterations of the cervical spinal column.

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loss under decompression are presented in the Appendix, two of whom recovered their hearing loss completely during recompression, and three who had only slight recovery. It seems likely that these five patients had sustained bubble formation in one or more branches of the internal acoustic artery. In the five patients not responding to recompression, but treated with decongestants, vasodilators, etc., the possibility also exists of microhemorrhage in the cochlea. A brief summary is given of current therapeutic regimens.

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## APPENDIX

### AUDIOLOGICAL NOTES ON SUDDEN DEAFNESS AMONG DIVERS

#### 1. C.R.A. EN1

Episode (1) Audiogram normal in 1963. Tank Instructor. On January 18, 1964, while not actually in the water, suffered severe loss of hearing in R ear, with some slight nausea. Two days later, audiometry showed a rather flat loss at 70-80 db. A dozen audiological tests (speech, recruitment, temporal integration, differential intensity and frequency discrimination, tone decay, Bekesy audiometry) indicated intracochlear peripheral organ involvement. Within three weeks hearing was normal except for a residual 15-db loss at 1 kc/s.

Episode (2) had been in water 45 min on October 14, 1965, up and down to 118 ft. Sudden disorientation of 90°, but no nausea. Within a few minutes he seemed normal, and made another 118-ft dive. Six days later he appeared for an audiogram, showing a U-shaped curve down to 30 db, within 6 more days back to previous acuity. No further episodes, though he subsequently broke the world free-dive record, but the 15-db dip at 1 kc/s remains today.

Impression: Two episodes of hemorrhage throughout the labyrinth, cause unknown.

#### 2. S.

In early 1968, on a dive to 350 ft for 30 min on HeO<sub>2</sub>, patient had fluid in middle ear and hearing loss. Placed on decongestants; 2-3 days later audiometry showed U-shaped at 70 db through 3 kc/s, up to 40 db at 8 kc/s. Four days later recovered to 60 db at 1-4 kc/s, and 1 yr later still U-shaped, down 45 db at 1-2 kc/s.

#### 3. W.R.L. SFC

450-ft dive on 28-31 January 1969. Pain in knees during decompression; two hrs after compression, decompression, and surfacing, noted mild hearing loss in R ear with tinnitus. Vasodilators and recompression were given. Irregularly flat audiogram loss, 50 db at 1 kc/s of which 5-10 db only recovered later.

#### 4. D.W.W. HMC

Same dive as W.R.L. At 56 ft, pt noted hearing loss in R ear. Some fluids in middle ear; decongestants were given. At 36 ft, tinnitus occurred, and lowered hearing. Recompression was given, but flat loss of 70 db continued, with little subsequent recovery.

#### 5. J.R.E.

Sudden profound loss of hearing at 110 ft decompression stop. Partly recovered during recompression at 165 ft after 5 min of breathing 36% O<sub>2</sub>. Diagnosis by Captain TAYLOR, ENT Clinic, Bethesda Naval Hospital, as "bends involving left internal acoustic artery." Residual U-shaped audiometric defect, maximum of 30 db at 500 c/s.

#### 6. J.H.G. HM1 (DV)

At 40 ft during decompression, noted sudden but moderate loss in R ear. Recompression to 60 ft, and later slow decompression, pt felt hearing improve back to normal.

7. K.C.B.

After 11 min at 95-ft stop during decompression, pt noted sudden loss in R ear, confirmed by low and high frequency tuning forks. Recompression to 165 ft brought acuity to normal after 15 min.

8. T.

Late 1966, in air compression chamber. He reached down to pick up something, and felt something happen to his L ear. One hour later, audiogram showed flat 50-db loss bilaterally. Only slight improvement after 4 mo, and loss persists to this date.

9. B.

Much like patient No. 5, JARVI; episode at first of low-tone loss during decompression, treated by recompression but with deficit of 70-80 db remaining.

10. M.

Episode (1) January 1968—had vertigo only during 350-ft dive.

Episode (2) June 1968—at 30-40 ft on HeO<sub>2</sub>, suffered unilateral profound acuity loss. On vasodilators, back to normal over 27 days, but tinnitus remains.

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13. ABSTRACT			
<p>Among those who are exposed repeatedly to compression-decompression cycles, a relatively high incidence of otologic symptoms including hearing loss is often reported. Most of this can readily be attributed to the residual effects of repeated aerotitis media, and is not sudden. A review of the topic of sudden deafness reveals that every large hospital where such records have been published sees every month about 1-2 patients whose sudden deafness is not easily explained. Possible causes have been suggested: acute neuritis of the VIIIth nerve, virus infection, vascular accident, vasomotor neurosis, acoustic trauma at levels of noise not usually noxious, collagen disease, transient ischemia from violent exercise or strong emotion, or from alterations of the cervical spinal column.</p> <p>None of these causes, however, would seem to underlie a series of patients experiencing sudden hearing loss of a moderate or often profound level during decompression, and which may respond favorably to immediate recompression. Five patients with hearing loss under decompression are presented in the Appendix, two of whom recovered their hearing loss completely during recompression, and three with only slight recovery. It seems likely that these patients had sustained bubble formation in one or more branches of the internal acoustic artery. In the others not responding to recompression, but treated with decongestants, vasodilators, etc., the possibility also exists of microhemorrhage in the cochlea. A brief summary is given of current therapeutic regimens.</p>			

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## KEY WORDS

## LINK A

## LINK B

## LINK C

ROLE

WT

ROLE

WT

ROLE

WT

Diver's hearing loss

Decompression hypacusis.